The Salient Points and The Value of Venous Angiocardiography in the Diagnosis of the Cyanotic Types of Congenital Malformations of the Heart

The Salient Points and the Value of Venous Angiocardiography In the Diagnosis of the Cyanotic Types of Congenital Malformations of the Heart

A TEN YEAR STUDY OF 421 ANGIOCARDIOGRAMS DONE ON 283 PATIENTS

By

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PREFACE

This book is the result of a ten year study of 121 angiocardiograms, each one consisting of approximately twenty films, taken on 283 patients with cyanotic types of congenital malformations of the heart.* The salient points in the angiocardiographic diagnosis of ten entities are presented.

In order to best establish the value of angiocardiography as a separate laboratory tool, the authors based the angiocardiographic diagnosis of each entity only on the knowledge that the patients were cyanotic, and on the interpretation of the angiocardiographic findings.

The accuracy of this angiocardiographic diagnosis was then checked in each case against the final diagnosis arrived at by a complete physical, fluoroscopic, roentgenologic and electrocardiographic examination and, in addition, by cardiac catheterization and/or surgery and/or autopsy.

Twenty-one entities of the cyanotic type of congenital malformations of the heart are classified into four groups. We wish to emphasize that this manuscript represents only a summary of the basic findings of the most important types of congenital malformations of the heart.

The authors wish to extend their thanks and appreciation to Dr. Charles T. Dotter, Professor and Chairman, Department of Radiology of the University of Oregon Medical School, for reading the proofs and offering many helpful suggestions. We also wish to thank our chief technician, Mr. Glen Volz and our artist, Mrs. Gloria Jones who have faithfully assisted in this work since its inception.

* The total number of angiocardiograms that the authors have studied on proven cases of various cyanotic and non cyanotic types of congenital malformations of the heart is over 1300.

CONTENTS

The Salient Points and The Value of Venous Angiocardiography in the Diagnosis of the Cyanotic Types of Congenital Malformations of the Heart

The following Key is for the drawings on pages 8, 17, 21, 28, 36, 44, 45, 51, 58, 61, 65

KEY


Venous blood


Arterial blood


Mixed blood predominantly arterial


Mixed blood predominantly venous

S V C —Superior Vena Cava
I V C —Inferior Vena Cava
R A —Right Atrium
R V —Right Ventricle
P A —Pulmonary Artery
P V —Pulmonary Veins
L A —Left Atrium
L V —Left Ventricle
A O —Aorta
C T —Common Trunk
Br A —Bronchial Artery

The figures in the cardiac chambers and in the vessels denote the respective percentage of their oxygen saturation

INTRODUCTION

THE PURPOSE of this book is to bring out the salient points in the angiocardiographic diagnosis of the various cyanotic types of congenital malformations of the heart and to establish the value of angiocardiography as a diagnostic tool for these entities. We, therefore, based our diagnosis on angiocardiographic findings alone and the knowledge that the patients were cyanotic. In order to best establish the diagnostic value of angiocardiography as a separate laboratory tool, all other available information regarding the history, physical, fluoroscopic, roentgenologic, electrocardiographic, cardiac catheterization and autopsy findings was purposely omitted. During the past ten years 421 angiocardiograms, each consisting of an average of twenty films, were done on 283 patients with cyanotic types of congenital malformation of the heart. Ninety five percent of our patients were under fifteen years of age. A complete physical, fluoroscopic, roentgenologic and electrocardiographic examination was done routinely on all patients before an angiocardiogram was performed. In addition to these examinations the diagnosis was confirmed in 149 patients by cardiac catheterization, in 101 patients by surgery and in 98 patients by autopsy findings. As a result of this study, we believe that the diagnostic value of angiocardiography can be best demonstrated by classifying the cyanotic types of congenital malformation of the heart into four groups and by the presentation of their salient angiocardiographic points.

*From the Hektoen Institute for Medical Research, the Pediatric Cardiophysiology Department of Cook County Children's Hospital, the Pediatric and Surgical Departments of the University of Illinois College of Medicine and of the Presbyterian Hospital of Chicago, Illinois. This study was aided by a grant from the Chicago Heart Association.

Group 1

Entities in which the diagnosis can *ALMOST ALWAYS* be made by proper interpretation of a technically good angiocardiogram (Table 1).

Group 2

Entities in which the diagnosis can *USUALLY* be made by proper interpretation of a technically good angiocardiogram (Table 2).

Group 3

Entities in which the diagnosis can *USUALLY NOT BE MADE* by proper interpretation of a technically good angiocardiogram (Table 3).

Group 4

Entities which *ALWAYS REQUIRE* additional studies before a definite diagnosis can be established (Table 4).

The salient points in the angiocardiographic diagnosis of the following ten cyanotic types of congenital malformations of the heart which belong to our Groups 1, 2 and 3 will now be presented briefly. Eleven other types of cyanotic congenital malformations of the heart belonging to Group 4, which represent entities where the value of angiocardiography alone is limited, will not be discussed.

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GROUP 1

TETRALOGY OF FALLOT

PATHOLOGY

1 Varying degrees of infundibular stenosis or combined infundibular and valvular stenosis Rarely, only valvular stenosis which is then usually associated with post-stenotic dilatation of the pulmonary artery Exceptionally, valvular or pulmonary atresia

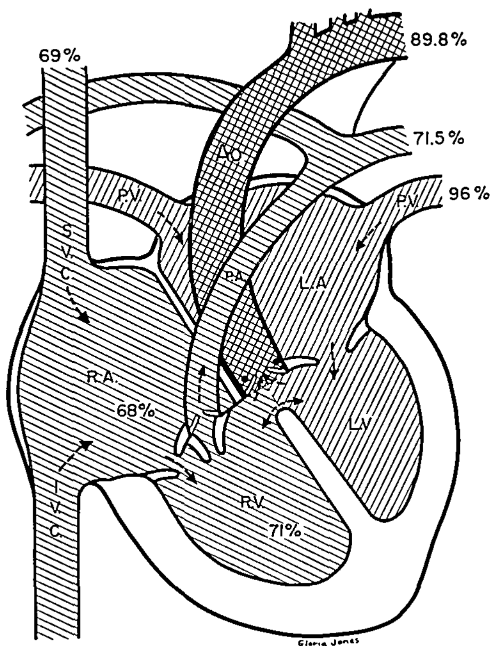
2 Large defect in the membranous and upper muscular portion of the ventricular septum

3 Varying degrees of functional dextroposition (overriding of aorta

4 Because of the above anomalies, the right ventricle is hypertrophied and, in some cases, dilated.

HEMODYNAMICS

Systemic venous blood returning to the right atrium and ventricle is diverted from the lung in proportion to the degree of obstruction caused by the stenosis. The venous blood which cannot enter the stenotic outflow tract will follow a path of lesser resistance and pass into the aorta This produces cyanosis. The systolic pressure in the right ventricle is approximately the same as in the left ventricle In all cases the pulmonary blood flow is decreased and the systemic flow is increased In cases where the pulmonary artery is atretic, the pulmonary circulation is maintained by bronchial arteries or other collateral vessels



TETRALOGY OF FALLOT

(See Key on page 2)

Salient Angiographic Features

1. Simultaneous visualization of the aorta and pulmonary arteries (Fig. 1-a).

2 In the majority of cases the main pulmonary artery and the branches are hypoplastic and the aorta is wide. In about 20% of these cases there is a right aortic arch (Fig. 1-b)

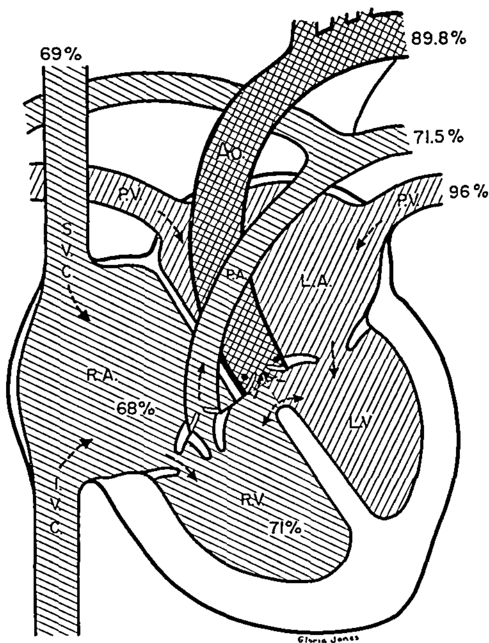
3. Patients with valvular or combined stenosis usually reveal a post-stenotic dilatation of the pulmonary artery.

4. In some patients with localized infundibular stenosis associated with valvular stenosis, there is opacification of a third ventricle. The size of this third ventricle varies depending upon the space between the localized infundibular stenosis and the pulmonary valves (Fig. 1-c).

5 Opacification of the peripheral pulmonary vessels is diminished.

6. Faint, or no opacification of the left cardiac chambers. Usually the fainter the opacification the more severe is the malformation. The opacification of the collateral vessels in these severe malformations is oftentimes well visualized (Fig. 1-d).

7 The type of stenosis (valvular, infundibular or combined) could at times not be determined in our series (Fig 1-e and 1-f).



TETRALOGY OF FALLOT

(See Key on page 2)

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7 The type of stenosis (valvular, infundibular or combined) could at times not be determined in our series (Fig. 1-e and 1-f).



Figure 1-a. Tetralogy of Fallot A-P view of a typical severe tetralogy of Fallot at 1½ seconds. Early and simultaneous visualization of hypoplastic pulmonary arteries and of a markedly dextroposed wide aorta. Note the location of the pulmonary semilunar valves (arrow), the opacification of the peripheral pulmonary vessels is diminished MPA—main pulmonary artery; LPA—left pulmonary artery, AO—aorta

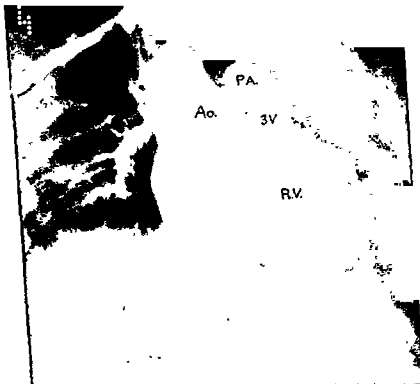


Figure 1-b A-P view at 2-seconds. Simultaneous visualization of pulmonary arteries and of a wide aorta with a right aortic arch. RV-right ventricle, 3V third ventricle, PA-pulmonary artery, AO aorta.

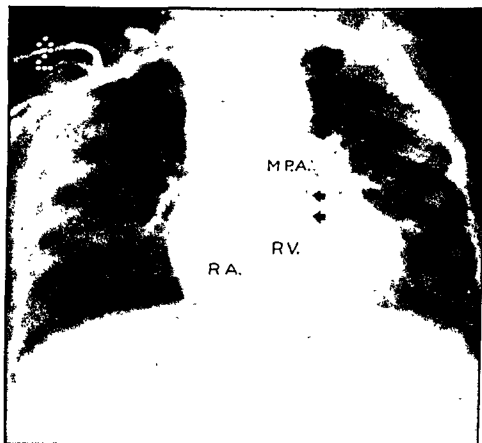


Figure 1-c. A-P view at 1-second. Simultaneous visualization of a wide aorta with a left aortic arch, and of the pulmonary arteries The infundibulum is very narrow (lower arrow), and the pulmonary semilunar valves are visualized (upper arrow). The third ventricle is seen between the two arrows. RA-right atrium, RV-right ventricle, MPA-main pulmonary artery.



Figure 1-d. A P view at 5½ seconds Severe tetralogy of Fallot with faint opacification of the left cardiac chambers. The reticulated appearance of the peripheral lung fields is due to marked collateral circulation.

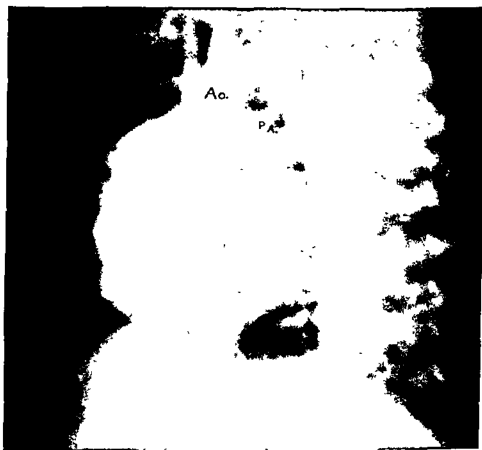


Figure 1-e LAO view at 1½ seconds. Note the overriding of the aorta partially from the opacified right ventricle and partially from the non-opacified left ventricle There is simultaneous visualization of markedly hypoplastic pulmonary arteries AO-aorta, P.A-pulmonary artery.



Figure 1-f RAO view at 2 seconds Note the stenotic infundibulum of the right ventricle which persisted throughout several cardiac cycles and is, therefore, a sign of generalized infundibular stenosis, and is not due to a momentary infundibular contraction in systole. There is an absence of the inferior vena cava and the hepatic veins enter the right atrium I-infundibulum, AO-aorta, AzV-azygos vein; H.V-hepatic vein.

COMPLETE TRANSPOSITION OF THE GREAT VESSELS

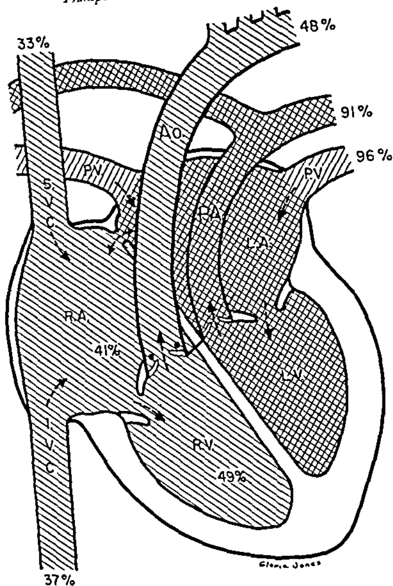
PATHOLOGY

1. Aorta originates from the right ventricle. Pulmonary artery from the left ventricle. Rarely, pulmonary stenosis may be present.

2. Associated defects in the form of atrial or ventricular septal defects or patent ductus arteriosus or anomalous entrance of pulmonary veins into the systemic veins or right atrium may be present.

HEMODYNAMICS

Venous blood enters the aorta from the right ventricle and oxygenated blood is pumped to the pulmonary circuit from the left ventricle. Life can only be maintained if associated defects that permit the mixing of venous and oxygenated blood are present. The more defects there are, the longer life is prolonged.



COMPLETE TRANSPOSITION OF ARTERIAL TRUNKS

(See Key on page 2)

Salient Angiographic Features

1. Left anterior oblique is the best view for the demonstration of an anteriorly displaced, densely opacified ascending aorta and of a very wide aortic arch (Fig. 2-a).

2. Absence of, or faint opacification of the pulmonary arteries at the time of the opacification of the aorta is characteristic.

3. Associated lesions such as septal defects or patent ductus arteriosus may at times be demonstrated (Fig. 2-b).

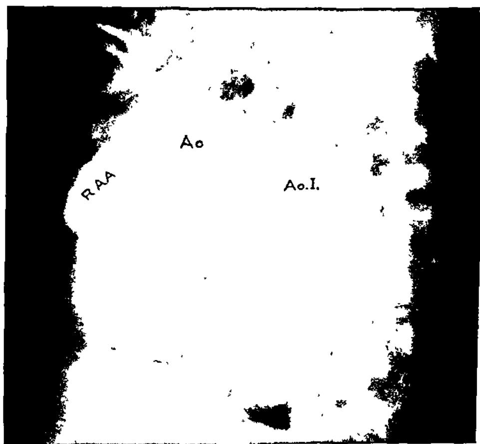


Figure 2-a. Transposition of the Great Vessels. LAO view at ½ second Note the size of the ascending aorta arising from the right ventricle and the anteriorly displaced wide aortic arch. The pulmonary artery is not opacified The infundibulum of the aorta is visualized in the region of the patent ductus arteriosus. R A A.-*right atrial appendage*, AO aorta; Ao.I.-aortic infundibulum.

1 The area of the cardiac silhouette that is not opacified during right heart filling (that is the area of the left atrium and of the left ventricle) is surprisingly large in view of the cyanosis.

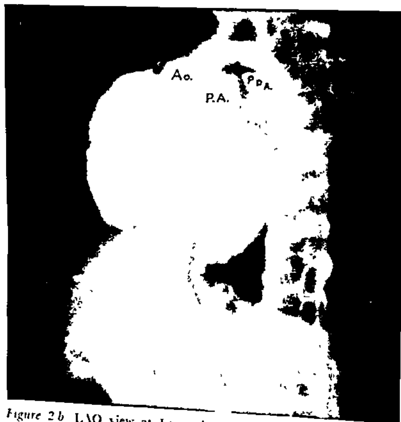


Figure 2 b LAO view at 1-second Opacification of the transposed aorta with a wide aortic arch. Visualization of the patent ductus arteriosus and of the pulmonary artery AO-aorta, PDA-patent ductus arteriosus, PA pulmonary artery

ANOMALOUS DRAINAGE OF ALL PULMONARY VEINS INTO THE SUPRACARDIAC SYSTEMIC VEINS

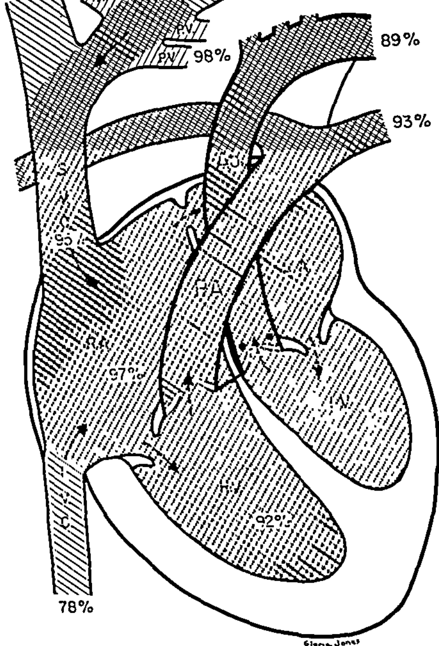
PATHOLOGY

1. All pulmonary veins enter into either the persistent left superior vena cava, left innominate vein, coronary sinus, right atrium, right superior vena cava, portal vein or ductus venosus

2. Associated communications between the cardiac chambers may be present.

HEMODYNAMICS

The right atrium receives not only the venous blood, but also all the oxygenated blood returning from the lungs. Unless there are associated lesions present, death takes place upon closure of the foramen ovale.



ANOMALOUS ENTRANCE OF ALL PULMONARY VEINS

(See Key on page 2)

Salient Angiographic Features

1. The pathognomonic sign is the early visualization of a markedly widened superior vena cava (Fig. 3-a) and its tributaries, followed first by the disappearance of the contrast substance (Fig. 3-b), and later by the reopacification of the above mentioned wide vessels (Fig. 3-c). This, of course, is only pathognomonic if there is no delayed reinjection of the contrast substance.

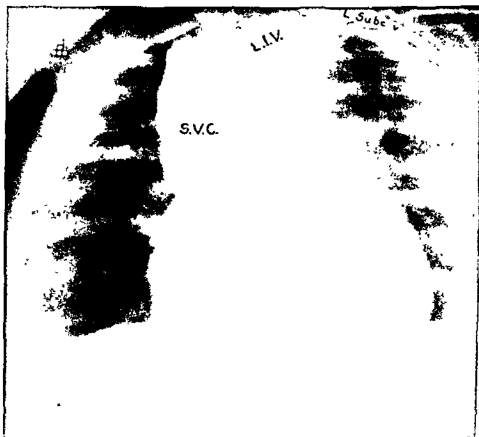


Figure 3-a. Anomalous entrance of all Pulmonary Veins. A-P view at ½ second. Marked dilatation of the right superior vena cava and of the left innominate vein. L. Subc. V-left subclavian vein; LIV-left innominate vein; SVC-superior vena cava.

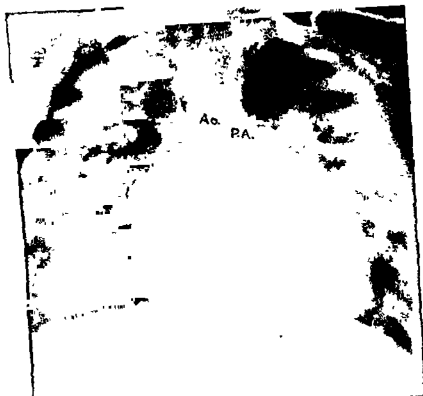


Figure 3 b A-P view of same angiocardiogram at two seconds Disappearance of the contrast substance from the superior vena cava and opacification of the right atrium, right ventricle and of the pulmonary arteries AO aorta, PA-pulmonary artery.

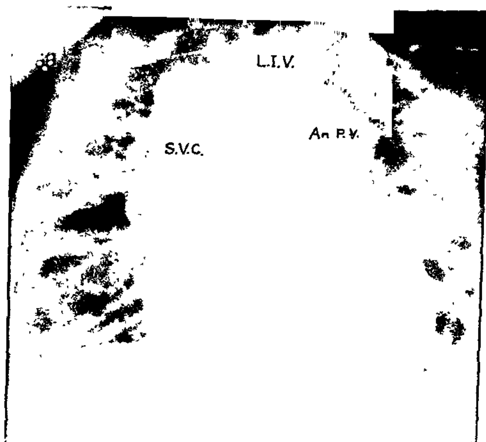


Figure 3-c. A-P view of same angiocardiogram at three seconds. Reappearance of the radio opaque substance into the left innominate vein and into the superior vena cava without reopacification of the left subclavian vein, thus excluding a delayed re-injection of the contrast substance. The presence of such reopacification is a pathognomonic sign of anomalous entrance of pulmonary veins into the great systemic veins. An. PV-anomalous pulmonary veins, LIV-left innominate vein, SVC-superior vena cava

GROUP 2

TRICUSPID ATRESIA

PATHOLOGY

1. Absence of the tricuspid valve
2. Patent foramen ovale or atrial septal defect
3. Hypoplastic or absent right ventricle
4. Pulmonary stenosis or atresia
5. Ventricular septal defect and/or patent ductus may be present
6. Uncommonly, tricuspid atresia may be associated with complete transposition of the great vessels. In these cases the pulmonary artery is usually wide, however, pulmonary stenosis may be present

HEMODYNAMICS

Venous blood from the right atrium must enter the left atrium and thence the left ventricle There is, therefore, always arterial oxygen unsaturation If there is no ventricular septal defect blood can enter the lung only through a patent ductus arteriosus or enlarged bronchial collateral vessels Unless there is an associated transposition of the great vessels with a large pulmonary artery originating from the left ventricle, pulmonary blood flow is always diminished

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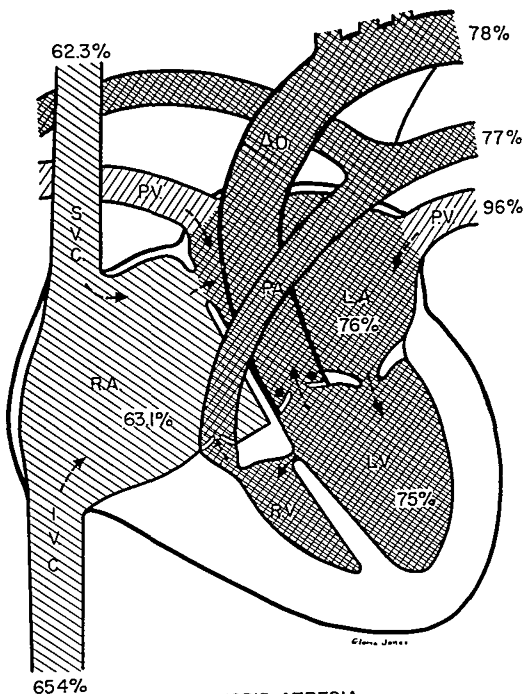
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TRICUSPID ATRESIA

(See Key on page 2)

Salient Angiographic Features

1 The best view is the left anterior oblique because there is no superimposition of the atria in this view (Fig. 4-a).

2 Immediate visualization of the shunt from right to left atrium (Fig. 4-b).

3. The opacification of the left atrium is usually intense. A part of the diaphragmatic surface of the heart in the A-P view usually occupied by the right ventricle may not be opacified in the early dextro angiocardiograms This filling defect becomes opacified in the later films if a ventricular septal defect is present as the contrast substance enters the hypoplastic right ventricle from the left ventricle. The pulmonary arteries are usually small and the appendage of the left atrium is seen to occupy the left upper medial border of the heart (Fig. 4-c).

4 There is an early dense opacification of a large left ventricle (Fig. 4 d)

5. The large right atrium and the counter-clockwise rotation of the heart due to the large left ventricle may simulate a dextrocardia (Fig. 4-e).

6 In cases of tricuspid atresia that are associated with complete transposition of the great vessels, the pulmonary artery originating from the large left ventricle is usually large and wide, and the aorta is hypoplastic However, pulmonary stenosis may be present either in the usual type of tricuspid atresia or in those with transposition of the great vessels



Figure 4-a. Tricuspid Atresia. LAO view at one second. Note shunt from right atrium into the left atrium and left ventricle. The location of the *atrial septal defect is well visualized (arrow) RA-right atrium,* LA-left atrium.

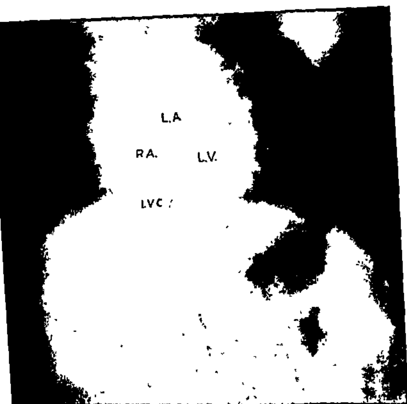


Figure 4-b LAO view at one second. Another LAO view of a tricuspid atresia with the contrast substance injected into the saphenous vein There is simultaneous visualization of the right atrium, left atrium and left ventricle. IVC-inferior vena cava, RA-right atrium; LA-left atrium, LV left ventricle



Figure 4-c. A-P view at one second Simultaneous visualization of the right atrium, left atrium, large left ventricle, aorta and hypoplastic pulmonary arteries. The area usually occupied by the right ventricle (arrow) is not opacified. Note visualization of the appendage of the left atrium. RA-right atrium, LV-left ventricle, LAA-left atrial appendage; AO-aorta.

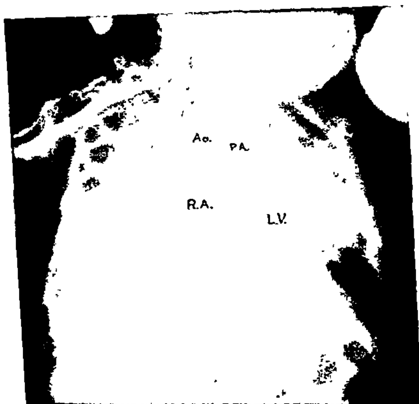


Figure 4-d. A P view at one second. Opacification of the large right atrium, left atrium, large left ventricle, hypoplastic pulmonary arteries and aorta RA right atrium, LV-left ventricle; PA pulmonary artery, AO-aorta

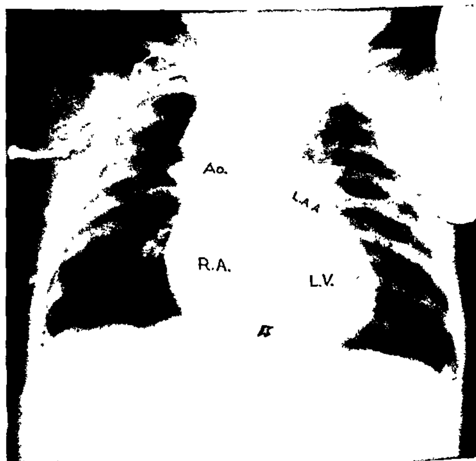


Figure 4-c A-P view at one second Simultaneous visualization of the right atrium, left atrium, large left ventricle, aorta and hypoplastic pulmonary arteries. *The area usually occupied by the right ventricle* (arrow) is not opacified. Note visualization of the appendage of the left atrium. RA-right atrium, LV-left ventricle, LAA-left atrial appendage, AO-aorta

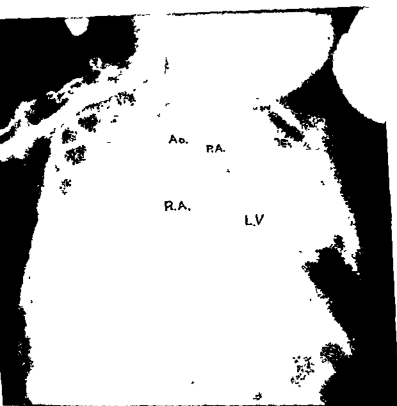


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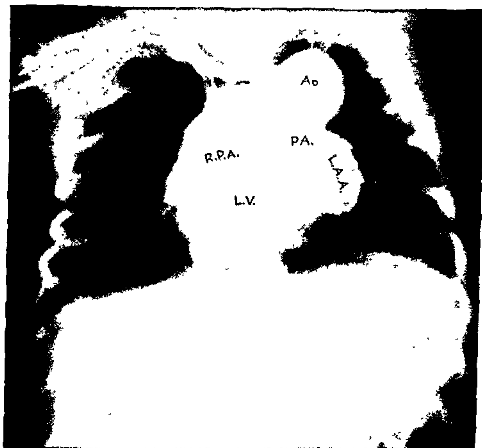


Figure 4-e. A-P view at 1½ seconds. Visualization of the large right atrium, left atrium and its appendage. The large aortic knob is seen on the upper left cardiac border. The area usually occupied by the right ventricle is not opacified and the pulmonary arteries are hypoplastic PA-pulmonary artery, RPA-right pulmonary artery, LAA-left atrial appendage; LV-left ventricle, AO-aorta.

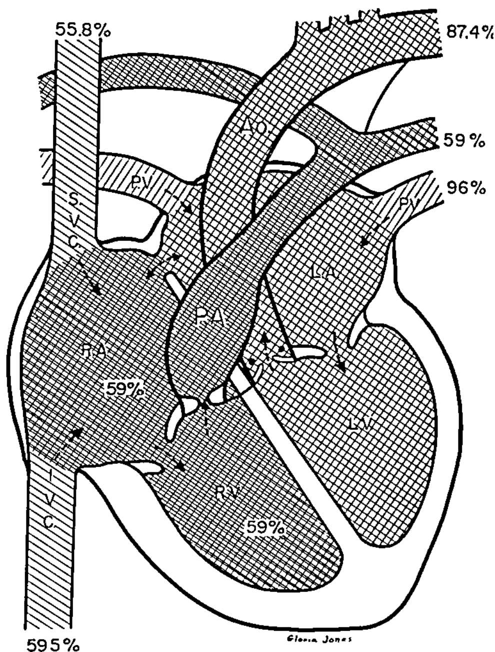
PULMONARY STENOSIS WITH NORMAL AORTIC ROOT AND ATRIAL COMMUNICATION AND RIGHT TO LEFT SHUNT

PATHOLOGY

1. Varying degrees of valvular stenosis in rare cases amounting to atresia. Usually post-stenotic dilatation of the pulmonary artery.
2. Patent foramen ovale or atrial communication.

HEMODYNAMICS

There is always right ventricular hypertension and low pulmonary arterial pressure Right ventricular hypertension results in an elevated right atrial pressure and a right to left shunt at the atrial level.



PULMONARY VALVULAR STENOSIS

(See Key on page 2)

Salient Angiographic Features

1. The right anterior oblique is the best view for the demonstration of the pulmonary arteries and valves (Fig 5-a).

2. An important diagnostic point is the very early opacification of the left atrium before the opacification of the aorta in the left anterior view (Fig 5-b). Since a portion of the contrast substance enters the right ventricle and the rest is shunted into the left atrium, the early opacification of the left atrium and of the left ventricle is not as intense as in patients with tricuspid atresia When the aorta is visualized it is not dextroposed (Fig 5-c).

3 In cases where there is a simultaneous visualization of the aorta and of the pulmonary artery, the presence of a post-stenotic dilatation of the pulmonary artery and of a definite enlargement of the transverse diameter of the heart usually exclude tetralogy of Fallot or pentalogy.

4 Post-stenotic dilatation of the main pulmonary artery is almost always present

5 The characteristic sign is the visualization of the jet-like formation of the contrast substance through the dome-shaped stenotic valve With our technique this finding was not often demonstrated (Fig 5-d)

6 Prolonged opacification of the pulmonary artery with a well opacified aorta (Fig. 5-e).

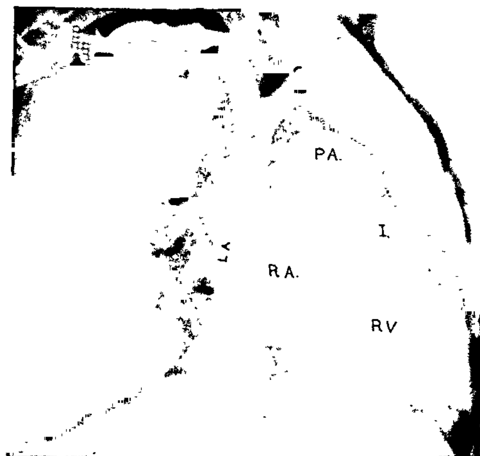


Figure 5-a Pulmonary Stenosis with Normal Aortic Root & Atrial Communication. RAO view at two seconds. Opacification of the right atrium, right ventricular inflow tract, the infundibulum and of the post-stenotic dilatation of the pulmonary artery. The right border of the left atrium can be visualized. RA-right atrium; RV-right ventricle, I-Infundibulum; PA-pulmonary artery; LA-left atrium.



Figure 5 b. LAO view at one second Case of severe valvular pulmonary stenosis with normal aortic root and atrial communication. Simultaneous opacification of large right atrium, right ventricle and left atrium Note shunt from right into left atrium (arrow) RA right atrium, LA left atrium

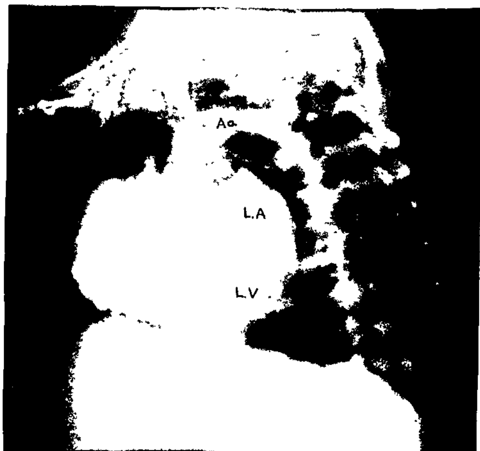


Figure 5-c. LAO view of same case at two seconds Visualization of right atrium, right ventricle, left atrium, left ventricle, hypoplastic pulmonary arteries and aorta The aorta is not dextroposed. LA-left atrium; LV-left ventricle, AO-aorta

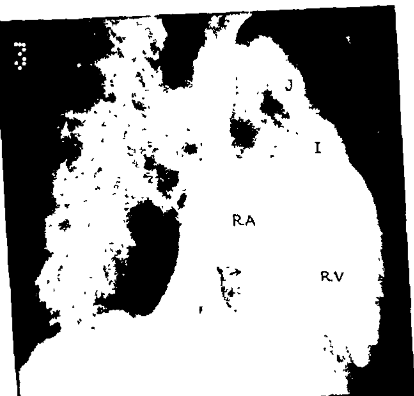


Figure 5 d RAO view at 1½ seconds Valvular stenosis. Opacification of the superior vena cava, right atrium, right ventricle and infundibulum Note the location of the tricuspid valve (arrow), the dome shaped area of the pulmonary valves and the jet-like propulsion of the contrast substance into the post-stenotically dilated pulmonary artery RA right atrium, RV-right ventricle, I.-infundibulum, J.-jet

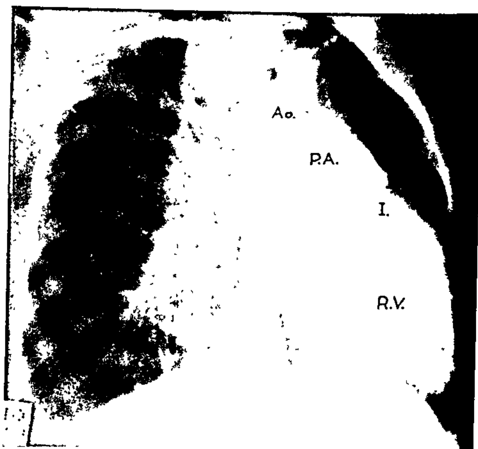


Figure 5-e. RAO view at 2½ seconds Same case as Figure 5-a. Opacification of right atrium, right ventricle, post-stenotic dilatation of the pulmonary artery and aorta RV-right ventricle, I-infundibulum, PA-pulmonary artery; AO-aorta.

PERSISTENT TRUNCUS ARTERIOSUS

PATHOLOGY

1. Two types are seen.

Type I. Common vessel arises from right and left ventricles. One or two large pulmonary arteries originate from the ascending portion of the common vessel.

Type II. One or two hypoplastic pulmonary arteries or no pulmonary arteries arise from the common trunk.

2. A high ventricular septal defect is present in both types.

HEMODYNAMICS

Type I. Mixed venous and arterial blood from both ventricles enters the common vessel. The pressure in both ventricles is about equal. The pulmonary circuit receives its blood supply directly from the common vessel under high pressure through the pulmonary arteries. Peripheral arterial oxygen unsaturation is always present, but because of the large volume of blood entering the lungs, this cyanosis may not be evident clinically.

Type II. The hemodynamics are the same as in Type I, except that much less blood enters the lung under decreased pressure, either from the small pulmonary arteries or from bronchial collateral vessels. Cyanosis is intense.

TYPE 1

With either one or two large pulmonary arteries coming off the common trunk.

TYPE 2

With either one or two small pulmonary arteries coming off the common trunk. Rarely, there may be no pulmonary arteries present. The angiocardiographic findings are entirely different in these two types.

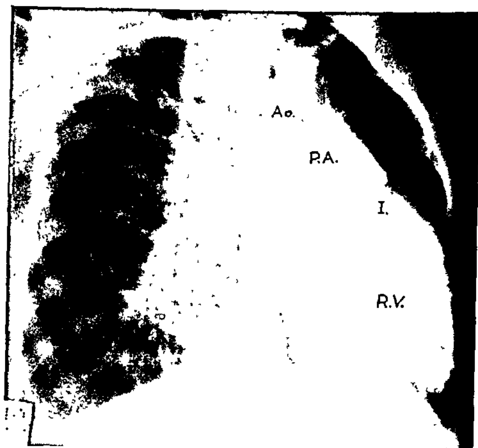


Figure 5-e RAO view at 2½ seconds Same case as Figure 5-a. Opacification of right atrium, right ventricle, post-stenotic dilatation of the pulmonary artery and aorta RV-right ventricle, I.-infundibulum, PA-pulmonary artery, AO-aorta

PERSISTENT TRUNCUS ARTERIOSUS

PATHOLOGY

1. Two types are seen.

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HEMODYNAMICS

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Type II. The hemodynamics are the same as in Type I, except that much less blood enters the lung under decreased pressure, either from the small pulmonary arteries or from bronchial collateral vessels. Cyanosis is intense.

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With either one or two small pulmonary arteries coming off the common trunk. Rarely, there may be no pulmonary arteries present. The angiocardiographic findings are entirely different in these two types.

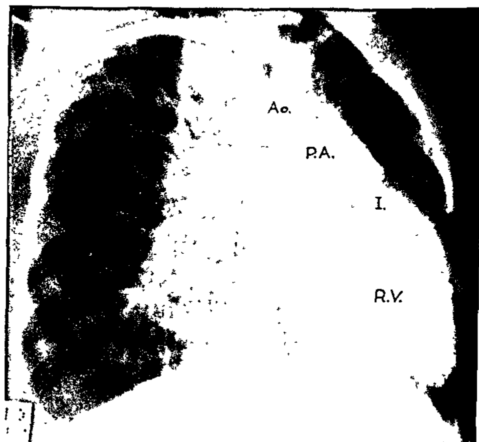


Figure 5-e. RAO view at 2½ seconds Same case as Figure 5-a Opacification of right atrium, right ventricle, post-stenotic dilatation of the pulmonary artery and aorta. RV-right ventricle, I-infundibulum, PA-pulmonary artery, AO-aorta.

PERSISTENT TRUNCUS ARTERIOSUS

PATHOLOGY

1. Two types are seen.

Type I. Common vessel arises from right and left ventricles. One or two large pulmonary arteries originate from the ascending portion of the common vessel.

Type II. One or two hypoplastic pulmonary arteries or no pulmonary arteries arise from the common trunk.

2. A high ventricular septal defect is present in both types.

HEMODYNAMICS

Type I. Mixed venous and arterial blood from both ventricles enters the common vessel. The pressure in both ventricles is about equal. The pulmonary circuit receives its blood supply directly from the common vessel under high pressure through the pulmonary arteries. Peripheral arterial oxygen unsaturation is always present, but because of the large volume of blood entering the lungs, this cyanosis may not be evident clinically.

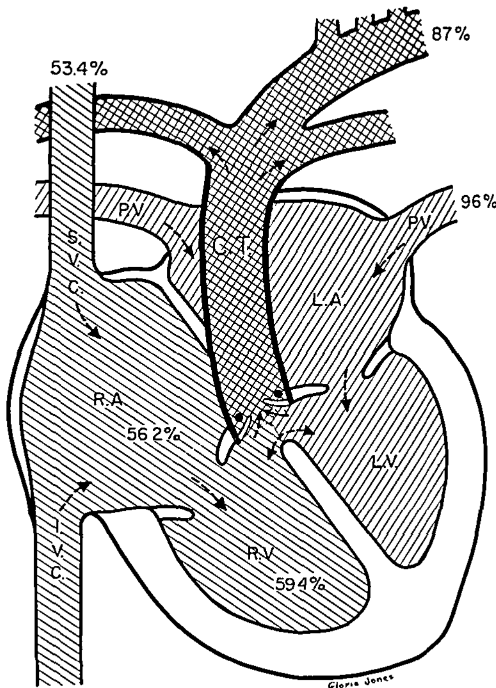
Type II. The hemodynamics are the same as in Type I, except that much less blood enters the lung under decreased pressure, either from the small pulmonary arteries or from bronchial collateral vessels. Cyanosis is intense.

TYPE 1

With either one or two large pulmonary arteries coming off the common trunk.

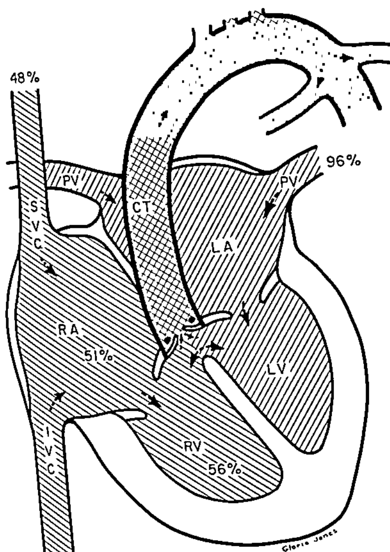
TYPE 2

With either one or two small pulmonary arteries coming off the common trunk. Rarely, there may be no pulmonary arteries present. The angiocardiographic findings are entirely different in these two types.



TRUNCUS COMMUNIS—Type I

(See Key on page 2)



TRUNCUS COMMUNIS—TYPE 2

Salient Angiographic Features

TYPE 1

1. In the A-P view there is usually a concavity in the pulmonary area because the pulmonary arteries originate from the trunk and, therefore, are in a higher position than normal. In addition, one of the pulmonary arteries may be absent (Fig. 6-a).

2. The origin of the pulmonary arteries, as well as the overriding of the truncus arteriosus, can be best demonstrated in the left anterior oblique view (Fig. 6-b).

3 The pulmonary vessels are large and well opacified when present.

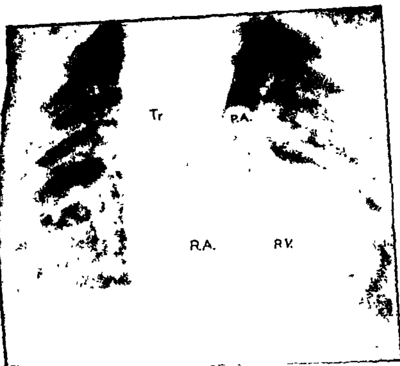


Figure 6-a *Persistent Truncus Arteriosus* A-P view at two seconds Visualization of right atrium, large right ventricle, very wide common trunk and large left pulmonary artery The concavity in the region of the pulmonary artery—a rather characteristic sign in the malformation—is thus seen to be due to the higher origin of the pulmonary artery from the common trunk and not from the outflow tract of the right ventricle R A-right atrium, RV right ventricle, Tr.-truncus communis, P A pulmonary artery.

Salient Angiographic Features

Type 1

1. In the A-P view there is usually a concavity in the pulmonary area because the pulmonary arteries originate from the trunk and, therefore, are in a higher position than normal. In addition, one of the pulmonary arteries may be absent (Fig. 6-a).

2. The origin of the pulmonary arteries, as well as the overriding of the truncus arteriosus, can be best demonstrated in the left anterior oblique view (Fig. 6-b).

3. The pulmonary vessels are large and well opacified when present.

Type 2

1. The difference between this type and type one is the finding of small pulmonary arteries and diminished opacification of the peripheral pulmonary vessels (Fig 6-c).

2. This diagnosis can only be made if the small pulmonary arteries are seen to emerge from the truncus arteriosus. Usually this type cannot be distinguished from an extreme tetralogy of Fallot.

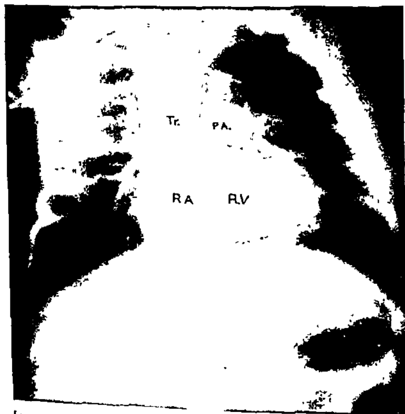


Figure 6 c AP view ... common trunk and a ... emerging from the ... RA-right atrium, RV-right ventricle, PA-pulmonary artery, Tr-truncus communis.

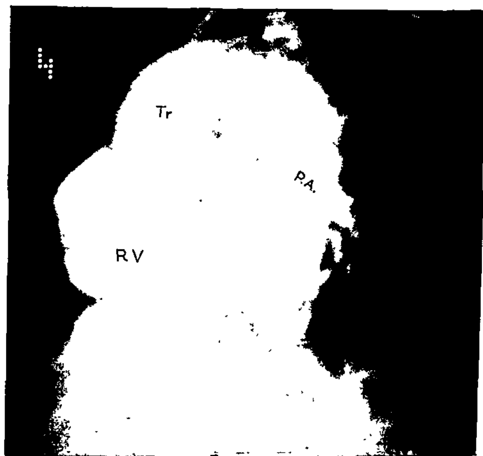
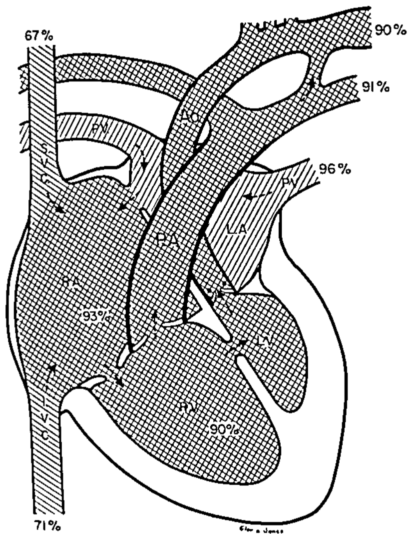


Figure 6-b LAO view at two seconds. Note the opacification of the right atrium, right ventricle and overriding wide common trunk. The pulmonary arteries are seen to originate from the common trunk. RV-right ventricle; PA-pulmonary artery, Tr.-truncus communis.



MITRAL ATRESIA

(See Key on page 2)

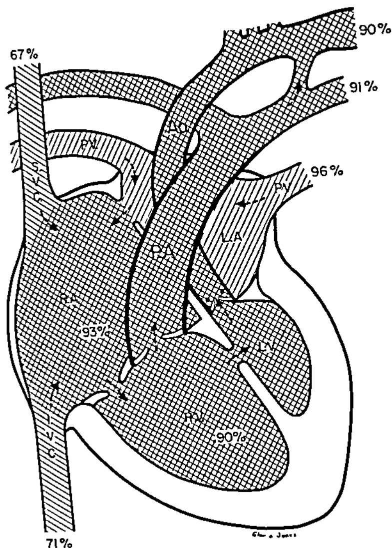
MITRAL ATRESIA WITH VENTRICULAR SEPTAL DEFECT

PATHOLOGY

1. Absence of mitral valve
2. Patent ductus arteriosus
3. This entity is always associated with the presence of one or more of some of the following additional defects: Atrial septal communication, ventricular septal defect, hypoplasia or atresia of the ascending aorta and of the aortic arch, single ventricle or hypoplasia of the left ventricle and anomalous return of the pulmonary veins.

HEMODYNAMICS

There is always arterial oxygen unsaturation. The oxygenated blood from the left atrium is shunted through an atrial communication into an enlarged right atrium, right ventricle, pulmonary artery and patent ductus arteriosus into the aortic arch and descending aorta. Unless there are associated lesions in the form of a ventricular septal defect or anomalous entrance of pulmonary veins into the systemic veins or right atrium, death takes place in early infancy.



MITRAL ATRESIA

(See Key on page 2)

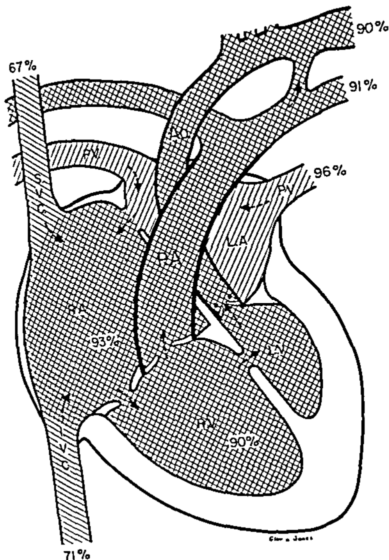
MITRAL ATRESIA WITH VENTRICULAR SEPTAL DEFECT

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MITRAL ATRESIA

(*See Key on page 2*)

Salient Angiographic Features

1. The left anterior oblique view is the best view and, so far as we know, angiocardiography is the only laboratory test to help establish the diagnosis (Fig. 7-a).

2. The most important angiocardiographic finding is the opacification of a hypoplastic left sided chamber in the presence of a large right atrium, right ventricle and pulmonary arteries (Fig 7-b).

3. Patent ductus may be visualized and the descending aorta may be wider and opacified to a greater degree than the proximal aorta.

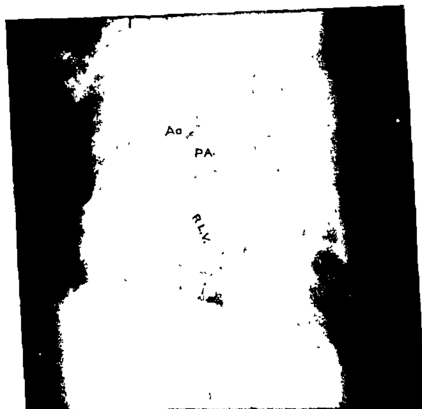


Figure 7-a Mitral Atresia LAO view at two seconds Simultaneous visualization of the right atrium, right ventricle, large pulmonary artery and aorta. Note the rudimentary left sided, posteriorly located chamber which communicates with the right ventricle through a ventricular septal defect. AO aorta, PA-pulmonary artery, RLV-rudimentary left ventricle

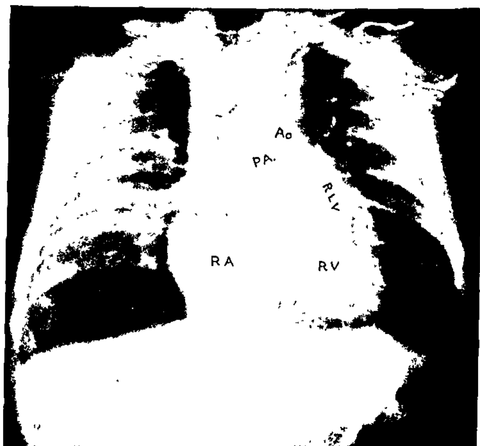


Figure 7-b. P-A view at one second Simultaneous visualization of the right atrium, right ventricle, pulmonary artery, rudimentary left ventricle and aorta. RA-right atrium, RV-right ventricle, PA-pulmonary artery; RLV-rudimentary left ventricle, AO-aorta

GROUP 3

TAUSSIG-BING ANOMALY

PATHOLOGY

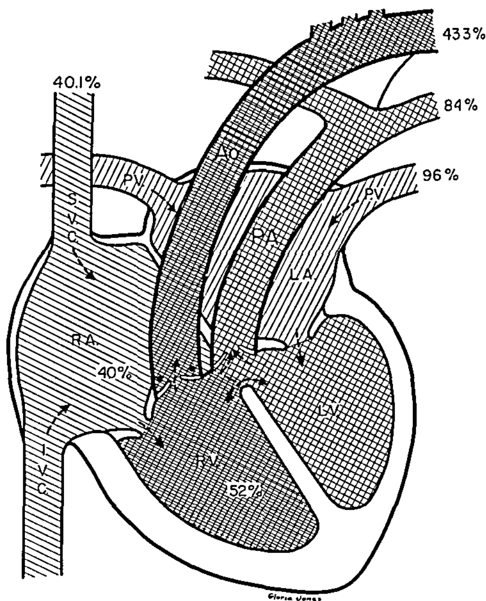
1. Transposition of the aorta.

2. Levoposition of the pulmonary artery, i.e., the pulmonary artery overrides both ventricles.

3. Ventricular septal defect of the membranous and upper muscular portion of the ventricular septum.

HEMODYNAMICS

Venous blood enters the aorta from the right ventricle. Venous blood from the right ventricle and arterialized blood from the left ventricle enter the pulmonary circulation through the overriding pulmonary artery. Pressures in both ventricles and both great vessels are approximately equal. Patency of the ductus arteriosus and the size of the ventricular defect, as well as the pulmonary resistance, determine the amount of blood that is shunted from the systemic into the pulmonary circuit and vice versa.



TAUSSIG-BING HEART

(See Key on page 2)

Salient Angiographic Features

1. Simultaneous visualization of an anteriorly transposed aorta and levoposed large pulmonary artery, best seen in the oblique views (Fig. 8 a).

2. The branches of the pulmonary arteries are usually well opacified.

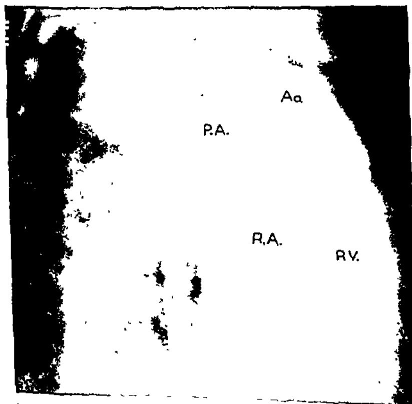


Figure 8-a Taussig-Bing Heart RAO view at one second Simultaneous visualization of the transposed aorta and of the levoposed large pulmonary artery RA right atrium, RV-right ventricle, AO aorta, PA-pulmonary artery

EISENMENGER COMPLEX

PATHOLOGY

Type I. The "anatomic" Eisenmenger—the rare type.

1 Functional dextroposition of the aorta is present from birth on.

2 Ventricular septal defect of the membranous and upper muscular portion.

3. Large pulmonary artery.

Type II. The common type.

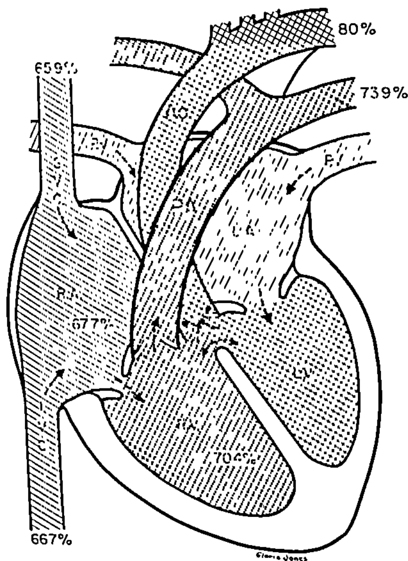
1. Large ventricular septal defect in the membranous portion of the septum. Right ventricular hypertrophy. Actual dextroposition of the aorta may be difficult to demonstrate even at autopsy.

2. Large pulmonary arteries.

HEMODYNAMICS

Type I. There is arterial oxygen unsaturation even though clinical cyanosis may not be evident, particularly early in life. The amount of venous blood shunted into the aorta depends upon the degree of functional dextroposition of the aorta and the height of the pulmonary resistance

Type II There is usually no arterial oxygen unsaturation for a number of years At first the shunt is from the left ventricle into the right one. For a number of years there is a gradual and progressive rise in the pulmonary resistance resulting in some of the blood from the right ventricle shunting into the aorta. Persistent cyanosis at rest is only present when the pulmonary resistance becomes higher than the systemic resistance. The degree of cyanosis depends upon the amount of blood shunted into the aorta



EISENMENGER COMPLEX

(See Key on page 2)

EISENMENGER COMPLEX

PATHOLOGY

Type I. The "anatomic" Eisenmenger—the rare type.

1. Functional dextroposition of the aorta is present from birth on.

2. Ventricular septal defect of the membranous and upper muscular portion.

3. Large pulmonary artery.

Type II. The common type.

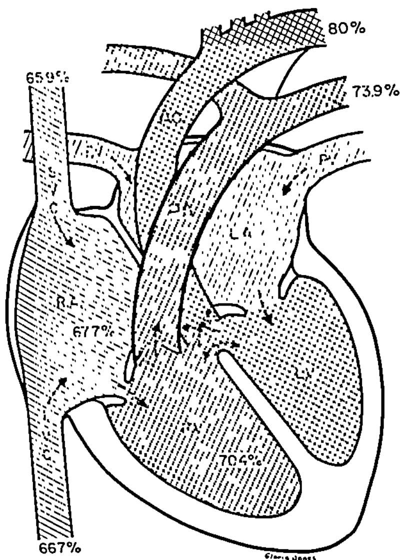
1. Large ventricular septal defect in the membranous portion of the septum. Right ventricular hypertrophy. *Actual dextroposition of the aorta may be difficult to demonstrate even at autopsy.*

2. Large pulmonary arteries.

HEMODYNAMICS

Type I. There is arterial oxygen unsaturation even though clinical cyanosis may not be evident, particularly early in life. The amount of venous blood shunted into the aorta depends upon the degree of functional dextroposition of the aorta and the height of the pulmonary resistance.

Type II. There is usually no arterial oxygen unsaturation for a number of years. At first the shunt is from the left ventricle into the right one. For a number of years there is a gradual and progressive rise in the pulmonary resistance resulting in some of the blood from the right ventricle shunting into the aorta. Persistent cyanosis at rest is only present when the pulmonary resistance becomes higher than the systemic resistance. The degree of cyanosis depends upon the amount of blood shunted into the aorta.



EISENMENGER COMPLEX

(*See Key on page 2*)

Salient Angiographic Features

1. Simultaneous visualization of a large pulmonary artery and aorta (Fig. 9-a).

2. The opacification of the pulmonary artery is usually greater than that of the aorta.

3. We divide the Eisenmenger's complex into two types:



Figure 9-a Eisenmenger's Complex A-P view at 3½ seconds Simultaneous visualization of large pulmonary arteries and of the aorta PA-pulmonary artery, AO-aorta

Type 1

Anatomic type with definite dextroposition of the aorta from birth on (Fig 9-b)

Type 2—The Common type

Development of the Eisenmenger syndrome in later life from a large ventricular septal defect This latter type cannot be diagnosed angiocardiographically before appearance of cyanosis, and the demonstration of a simultaneous visualization of the dextroposed aorta and of the large pulmonary arteries

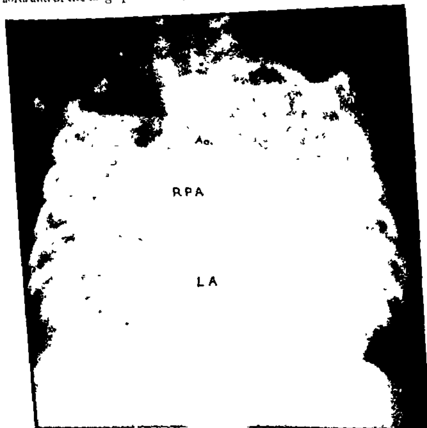


Figure 9-b AP view at 1½ seconds Simultaneous visualization of markedly enlarged pulmonary arteries and of a small aorta Note the aneurysmal dilatation of the right pulmonary artery RPA-right pulmonary artery LA left atrium, AO-aorta

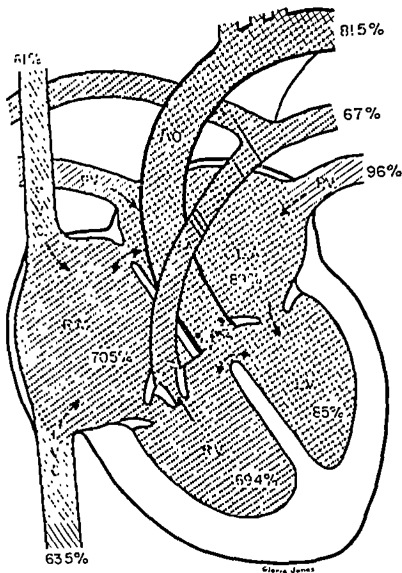
PENTALOGY

PATHOLOGY

1. Tetralogy of Fallot and an atrial communication in the form of a patent foramen ovale or an atrial septal defect.

HEMODYNAMICS

If there is no evidence of right heart failure and the diastolic pressure in the right ventricle is within normal limits, the hemodynamics are the same as in a tetralogy of Fallot. In the presence, however, of an atrial septal defect and normal diastolic pressure in the ventricle, the shunt will be from the left atrium into the right atrium If the diastolic pressure in the right ventricle is elevated, there will be a shunt from right atrium to the left atrium, irrespective of whether there is a patent foramen ovale or an atrial septal defect.



PENTALOGY (TETRALOGY AND ATRIAL COMMUNICATION)

(See Key on page 2)

Salient Angiographic Features

The presence of a right-to-left shunt at the atrial level makes the angiocardiographic diagnosis very difficult.

1. The left anterior oblique view is the best.

2. Early and, at times, simultaneous visualization of the right atrium, left atrium, right ventricle, pulmonary arteries and aorta (Fig. 10-a).

3. The left atrium is usually not as densely opacified as in tricuspid atresia. The aorta overrides both ventricles—a point of greatest importance in the differential diagnosis from pulmonary stenosis with interatrial communication and a normal aortic root (Fig. 10-a).

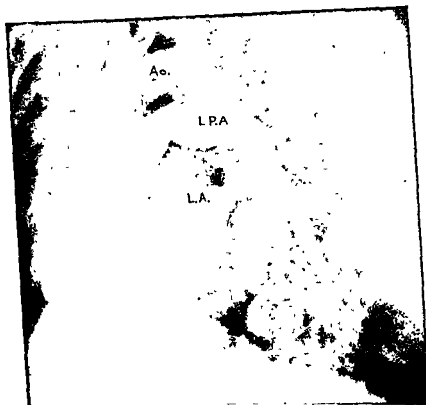


Figure 10 a. Pentalogy. LAO view at one second. Simultaneous visualization of the well opacified right atrium, right ventricle, pulmonary arteries and of the faintly opacified left atrium and dextroposed aorta. LA left atrium. LPA left pulmonary artery, AO aorta

DISCUSSION

Table 1 consists of the entities in which the diagnosis can almost always be made by proper interpretation of a technically good angiocardiogram; tetralogy of Fallot, transposition of the great vessels and anomalous drainage of the pulmonary veins into the systemic veins.

TETRALOGY OF FALLOT

Ninety-three patients were studied. Out of 127 angiocardiograms reviewed the diagnosis was correctly made in 96 (75.5%) The diagnosis was highly suggestive, but not made with certainty, and the possibility of another diagnosis was considered in 7 (5.5%). Only in 6 (4.7%) of our angiocardiograms was the diagnosis incorrectly made. Of the total number of angiocardiograms, 13 or 10.4% were technically poor and visualization of the various chambers of the heart and the great vessels was impossible, and 5 angiocardiograms (3.9%) were missing from our files The technically poor angiocardiograms were encountered in the earlier years of this study. It is thus clear that the percentage of the correct angiocardiographic diagnoses could have been about 90% if we add the 10.4% of the technically poor and 3.9% of the missing angiocardiograms to the total correctly diagnosed

Of the 93 patients studied the diagnosis of tetralogy of Fallot was confirmed by catheterization in fifty-six patients, by surgery in fifty patients and by autopsy in twenty-three cases

TRANSPOSITION OF THE GREAT VESSELS

Thirty-five patients were studied Out of 48 angiocardiograms, 40 or 83.3% were correctly diagnosed, and only in two cases (4.2%) was the diagnosis not definite. This group represents, therefore, even a higher percentage of correct angiocardiographic diagnoses than those of tetralogy of Fallot In the 2 cases where the diagnosis was not definite the angiocardiogram was taken in the A-P view Had they been taken in the left anterior oblique view—which we

TABLE 1

Entities in Which the Diagnosis Can *ALMOST ALWAYS* Be Made by Proper Interpretation of a Technically Good Angiocardiogram

Name of Entity	*Total Number*		*Percentage of Angiocardiographic Diagnosis*			*Improper Technique*	*Angiocardiograms not Available*	*Diagnosis Confirmed*		
	Patients	*Angiocardiograms*	*Established*	*Highly Suggestive*	*Not Established*			*Catheterization*	*Surgery*	*Post-Mortem Exam*
Tetralogy of Fallot	93	127	96 75.5%	7 5.5%	2 4.7%	13 10.4%	5 3.9%	56	50	23
Transposition of the Great Vessels	35	48	40 83.3%	2 4.2%		2 4.2%	4 8.3%	12	7	23
Anomalous Drainage of All Pulmonary Veins into Systemic Veins	5	6	5 83.33%			1 16.67%		4	1	3

DISCUSSION

Table I consists of the entities in which the diagnosis can almost always be made by proper interpretation of a technically good angiocardiogram; tetralogy of Fallot, transposition of the great vessels and anomalous drainage of the pulmonary veins into the systemic veins.

TETRALOGY OF FALLOT

Ninety-three patients were studied. Out of 127 angiocardiograms reviewed the diagnosis was correctly made in 96 (75.5%). The diagnosis was highly suggestive, but not made with certainty, and the possibility of another diagnosis was considered in 7 (5.5%). Only in 6 (4.7%) of our angiocardiograms was the diagnosis incorrectly made. Of the total number of angiocardiograms, 13 or 10.4% were technically poor and visualization of the various chambers of the heart and the great vessels was impossible, and 5 angiocardiograms (3.9%) were missing from our files. The technically poor angiocardiograms were encountered in the earlier years of this study It is thus clear that the percentage of the correct angiocardiographic diagnoses could have been about 90% if we add the 10 4% of the technically poor and 3 9% of the missing angiocardiograms to the total correctly diagnosed.

Of the 93 patients studied the diagnosis of tetralogy of Fallot was confirmed by catheterization in fifty-six patients, by surgery in fifty patients and by autopsy in twenty-three cases.

TRANSPOSITION OF THE GREAT VESSELS

Thirty-five patients were studied. Out of 18 angiocardiograms, 10 or 83.3% were correctly diagnosed, and only in two cases (4.2%) was the diagnosis not definite This group represents, therefore, even a higher percentage of correct angiocardiographic diagnoses than those of tetralogy of Fallot In the 2 cases where the diagnosis was not definite the angiocardiogram was taken in the A-P view. Had they been taken in the left anterior oblique view—which we

TABLE 2

ENTITIES IN WHICH THE DIAGNOSIS CAN *USUALLY* BE MADE BY PROPER INTERPRETATION OF A TECHNICALLY GOOD ANGIOCARDIOGRAM

Name of Entity	*Total Number*		*Percentage of Angiocardiographic Diagnosis*			*Improper Technique*	*Angiocardiograms not Available*	*Diagnosis Confirmed*		
	Patients	*Angiocardiograms*	*Established*	*Highly Suggestive*	*Not Established*			*Catheterization*	*Surgery*	*Post Mortem Exam*
1. Tricuspid Atresia	14	21	16 76.1%	4 19.1%			1 4.8%	5	12	4
2. Puomonary Stenosis with Normal Aortic Root and Atrial Communication and Right to Left Shunt	30	39	27 69.3%	7 17.9%	3 7.6%	2 5.1%		20	14	4
3. Persistent Truncus Arteriosus	16	28	20 71.4%	6 21.4%	1 3.6%		1 3.6%	7	4	8
4. Mitral Atresia with Ventricular Septal Defect	3	6	4 66.7%		2 33.3%			1	3	4

consider the best one for the demonstration of the transposition of the great vessels—the correct diagnosis could have been most probably made. The number of the technically poor and missing angiocardiograms was 2 (4.2%) and 4 (8.3%) respectively. The diagnosis in the case of these 35 was confirmed in twelve patients by catheterization, in seven patients by surgery and in twenty-three patients by post mortem examination.

ANOMALOUS DRAINAGE OF ALL PULMONARY VEINS INTO SUPRACARDIAC SYSTEMIC VEINS

Six angiocardiograms were taken on five patients. The angiocardiographic diagnosis could have been most probably as high as 100% were it not for one technically poor angiocardiogram. We wish to emphasize that we are considering here only the anomalous drainage of all pulmonary veins into the systemic veins, and not those cases where the anomalous veins drain directly into the right atrium or into the coronary sinus. In the latter, the percentage of correct angiocardiographic diagnoses is considerably lower. The diagnosis in this group of five patients was confirmed in four patients by catheterization, in one patient by surgery and in three patients by post-mortem examination.

Table 2 contains four entities in which the diagnosis can usually be made by proper interpretation of a technically good angiocardiogram; tricuspid atresia, pulmonary stenosis with normal aortic root and atrial communication, persistent truncus arteriosus and mitral atresia.

TRICUSPID ATRESIA

Twenty-one angiocardiograms taken on 11 patients have been studied. The diagnosis was made in 16 (76.2%) and was highly suggestive in 4 (19.1%). One angiocardiogram (4.9%) was missing. In 5 instances the diagnosis was confirmed by catheterization, in 12 by surgery and in 4 by post mortem examination.

Table 3 contains entities in which the diagnosis can usually not be made by proper interpretation of a technically good angiocardiogram: Taussig-Bing heart, Eisenmenger's complex and pentalogy

TAUSSIG-BING ANOMALY

Seventeen angiocardiograms done on eight patients were studied The diagnosis was correct in 8 (47.2%), and in 5 (29.4%) the diagnosis was highly suggestive. Two (11 7%) of the angiocardiograms are missing The diagnosis was confirmed by catheterization in six out of seven patients Because we believe that in the majority of cases it is impossible to differentiate with certainty angiocardiographically a Taussig-Bing heart from complete transposition of the great vessels with a large ventricular septal defect, and even at autopsy this differential diagnosis cannot always be made, we included in this group our patients with transposition of the great vessels with large ventricular septal defects in which the pulmonary artery was visualized simultaneously with the transposed aorta

EISENMENGER'S COMPLEX

Out of the 24 angiocardiograms done on sixteen patients 13 (54 9%) were correctly diagnosed The diagnosis was highly suggestive in 5 (20 8%), and was not made in 3 (12 5%) One (4 2%) angiocardiogram was not used because of poor technique Diagnosis has been confirmed in eight patients by catheterization, in one patient by surgery and in eight patients by post mortem examination

PENTALOGY

Twelve angiocardiograms were done on eight patients. The diagnosis was made in 7 (58 1%), was highly suggestive in 4 (33 3%) and was incorrectly made in 1 (8 3% The diagnosis was confirmed in four of the patients by catheterization, two by surgery, and on four patients by post mortem examinations.

These three entities belong to Group 3 because of the generally lower percentage of correctly diagnosed angiocardiograms, higher percentage of doubtful diagnosis and higher percentage of incorrect diagnosis than encountered in Group 1 and Group 2.

PULMONARY STENOSIS WITH NORMAL AORTIC ROOT AND ATRIAL COMMUNICATION AND RIGHT TO LEFT SHUNT

Out of 39 angiocardiograms taken on thirty patients 27 (69.3%) were diagnosed. In 7 (17.9%) the diagnosis was highly suggestive and in 3 (7.6%) diagnosis could not be established. Only two angiocardiograms (5.1%) were technically poor. The diagnosis was confirmed by catheterization in twenty patients, by surgery in fourteen patients and by post mortem examination in four patients. The doubtful diagnoses in this entity were due to the fact that the angiocardiograms were taken in the A-P view. Had these been taken in the oblique view where the shunt from right-to-left atrium can be best demonstrated, the diagnosis could have been made in a higher percentage. The need of a second angiocardiogram taken in the oblique view is thus demonstrated

PERSISTENT TRUNCUS ARTERIOSUS

Sixteen patients were studied, including the two types of persistent truncus arteriosus Out of the 28 angiocardiograms examined, 20 (71 4%) were diagnosed correctly, and in 6 (21 4%) the diagnosis was highly suggestive. A correct diagnosis was not established in only one patient (3 0%) The diagnosis was confirmed by catheterization in seven patients, by surgery in four patients, and by post mortem examination in eight patients

MITRAL ATRESIA WITH VENTRICULAR SEPTAL DEFECT

Three patients and 6 angiocardiograms were studied. The diagnosis was made in four angiocardiograms (66.6%). The diagnosis was confirmed in three patients by surgery, one by catheterization and in four patients by post mortem examination.

The above four entities belong to Group 2 instead of Group 1, because of the generally lower percentage of correct diagnosis and greater percentage of highly suggestive diagnosis

COMMENTS

Our own interpretations of our angiocardiograms have been constantly changing in the light of a correlative clinical, roentgenologic, electrocardiographic, catheterization, surgical and post mortem findings

The improvement in our techniques, such as the use of rectal barbiturate sedation for our infants and younger children, the very rapid injection of the contrast substance, the improved radiography, have all been very important factors in obtaining satisfactory visualization of the various cardiac chambers and of the great vessels and of their tributaries The results obtained in this study are based on our experiences in the use of venous angiocardiography We used a Fairchild camera and our films were taken at ½ second intervals at 1/60 second exposure time. The technique of venous angiocardiography is rather simple and is discussed fully in the standard works on angiocardiography by Drs Dotter and Steinberg, Dr. Castellanos and associates, and others This technique does not require the use of very expensive elaborate apparatus, and one physician and usually one technician are all that is necessary for this procedure Dr. Kjellberg and associates made extensive use of selective angiocardiography by injecting the contrast substance through a catheter as closely as possible into a particular area of the heart under investigation. This same group as well as Drs Wegelius and Lind, have utilized a specially constructed machine that enables them to take twenty-four films per second—twelve films in two different planes at right angles to each other and at an exposure time as fast as 0 003 seconds

Their contributions are most valuable and the many details that they were able to obtain, such as the exact location and size of the stenosis, the degree of the dextroposition of the aorta and

TABLE 3

ENTITIES IN WHICH THE DIAGNOSIS CAN *USUALLY NOT* BE MADE BY PROPER INTERPRETATION OF A TECHNICALLY GOOD ANGIOCARDIOGRAM

Name of Entity	*Total Number*		*Percentage of Angiocardiographic Diagnosis*			*Improper Technique*	*Angio-cardio-grams not Available*	*Diagnosis Confirmed*		
	Pa-tients	*Angio-cardio-grams*	*Estab-lished*	*Highly Suggestive*	*Not Estab-lished*			*Cathe-terization*	*Surgery*	*Post Mortem Exam.*
1 Taussig-Bing Anomaly	8	17	8 47.2%	5 29.4%		2 11.7%	2 11.7%	6		
2 Eisenmenger's Complexes	16	24	13 54.2%	5 20.8%	3 12.5%	1 4.2%	2 8.3%	8	1	8
3. Pentalogy	8	12	7 58.4%	4 33.3%	1 8.3%			4	2	4

CONCLUSIONS

A study of 121 angiocardiograms done on 283 patients led to the following conclusions regarding the diagnostic value of venous angiocardiography.

1) The diagnosis of tetralogy of Fallot, complete transposition of the great vessels and anomalous entrance of the pulmonary veins into the great systemic veins can *ALMOST ALWAYS* be made from a properly performed and properly interpreted angiocardiogram.

2) The diagnosis of tricuspid atresia, pulmonary stenosis with normal aortic root and atrial communication and right to left shunt, persistent truncus arteriosus and mitral atresia with ventricular septal defect can *USUALLY* be made from a properly interpreted angiocardiogram.

3) The diagnosis of Taussig-Bing anomaly, Eisenmenger's complex and pentalogy in which the diagnosis can *USUALLY NOT BE MADE* by proper interpretation of a technically good angiocardiogram.

4) All other forms of the cyanotic types of congenital malformations of the heart *ALWAYS REQUIRE ADDITIONAL* studies before a definite diagnosis can be established.

5) The salient angiocardiographic findings in ten entities are presented.

of the pulmonary artery, the location and the size of the septal defects, cannot be duplicated by venous angiocardiography, and by the use of a machine that takes only two films per second at 1/60 second exposure time. On the other hand, selective angiocardiography is a much more complicated and time consuming procedure. A general anesthetic, cardiac catheterization and a competent team of workers are necessary for this procedure. From our limited experience with selective angiocardiography, we believe that this angiocardiographic procedure is attended with greater danger to the patient. In this series of 421 angiocardiograms no death occurred directly attributable to the procedure. We wish to state that while we believe that in the vast majority of all cases, the experienced clinician can establish a clinical diagnosis without angiocardiography and/or catheterization, it is the experience gained from correlative studies of angiocardiography, catheterization and autopsy material combined with the history, physical, fluoroscopic, roentgenologic and electrocardiographic studies that make it possible for the clinician to establish these clinical diagnoses.

So far as we know no previous attempt to establish the value of angiocardiography as a separate laboratory tool has been published previously. We purposely omitted any references to the clinical, roentgenologic, electrocardiographic and cardiac catheterization findings in any of these entities Although our division of the cyanotic types of congenital malformations of the heart into 4 groups may appear to be an artificial one and although we realize that some authorities in this field will differ with us regarding this classification, the results recorded in this manuscript are those based on our personal experiences No attempt was made by us to minimize the percentages of the technically poor angiocardiograms as well as the percentage of those angiocardiograms which offered difficulties in arriving at a correct diagnosis

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TABLE 4

ENTITIES WHICH *ALWAYS REQUIRE* ADDITIONAL STUDIES BEFORE A DEFINITE DIAGNOSIS CAN BE ESTABLISHED*

Name of Entity	*Patients*	*Angio-cardiograms*
Congenital Mitral Stenosis	3	5
Tricuspid Stenosis	4	8
Tricuspid Atresia with Transposition of the Great Vessels	3	5
Tricuspid Atresia with Persistent Truncus Arteriosus	3	6
Single Ventricle	4	8
Ebstein's Anomaly	4	8
Levocardia	6	11
Isolated Dextrocardia	3	3
Pulmonary Stenosis with Ventricular Septal Defect	5	8
Pulmonary Stenosis with Patent Ductus Arteriosus	1	3
Cor Pulmonale	1	1
Patent Ductus Arteriosus with Reversal of the Shunt	3	3
Primary Pulmonary Hypertension	3	4
Anomalous Entrance of All Pulmonary Veins into Right Atrium	1	1
Cyanosis of Non-Cardiac origin	11	19
Total	55	93

* The authors realize that several of these entities particularly Patent Ductus Arteriosus with Reversal of the Shunt, could properly be placed in Table II or III.

This book

THE SALIENT POINTS AND THE VALUE OF VENOUS ANGIOCARDIOGRAPHY IN THE DIAGNOSIS OF THE CYANOTIC TYPES OF CONGENITAL MALFORMATIONS OF THE HEART

By

BENJAMIN M. GASUL, M.D., GERSHON HAIT, M.D., ROBERT F. DILLON, M.D.
and
EGBERT H. FELL, M.D.

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